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Changes in Intracardiac Atrial Cardioversion Threshold at Rest and During Exercise

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Objectives. **We sought to analyze in patients with chronic atrial fibrillation (AF) the change in the intracardiac atrial defibrillation threshold (ADT) at rest and during exercise, to quantify the effective risk of low energy endocavitary cardioversion during the effort and to compare the ADT of chronic and reinduced AF.**

Background. **Low energy endocavitary cardioversion is a new alternative to transthoracic shock in patients with chronic AF. Nevertheless, patient discomfort and possible induction of ventricular arrhythmias should be further evaluated.**

Methods. **Sixteen patients with chronic AF were included in the study. Two 6F custom-made catheters (Electro-Catheter, Inc.) were used for shock delivery and one tetrapolar lead for ventricular synchronization. Without sedation and in a random order, patients underwent two sequences of shocks to determine the ADT at rest and during exercise. Exercise was performed isometrically by the superior limbs. Atrial fibrillation was reinduced by atrial pacing. After each shock, the patients were requested to grade their discomfort with a score from 1 to 5. The power of the study was >90% in detecting a 25% difference in the ADT between groups.**

Results. **Patients were classified into two groups: Nine patients (group A) underwent the first cardioversion during exercise; seven patients (group B) underwent the first cardioversion at rest. In total, the mean (±SD) ADT was 6.70 ± 1.54 J during exercise and 7.02 ± 1.82 J at rest ($p = 0.59$). A significantly lower ADT was observed in the second shock sequence than the first one (6.32 ± 2.09 J vs. 7.40 ± 0.87 J, $p < 0.05$). The discomfort score was 3.25 ± 0.86 at rest and 2.94 ± 0.77 during exercise ($p = 0.09$). No complications occurred.**

Conclusions. **Low energy endocavitary cardioversion is a safe and effective procedure in patients with chronic AF. Discomfort is not generally severe enough to result in procedure termination. The ADT is not influenced by exercise and is higher in chronic than in reinduced AF.**

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Low energy intracardiac transcatheter cardioversion of paroxysmal and chronic atrial fibrillation (AF) has recently been proposed as an alternative to transthoracic cardioversion or as the last possible therapy for patients refractory to external atrial defibrillation (1–4). Furthermore, an implantable device for automatic or manual intracardiac atrial cardioversion will be soon available (5,6), giving rise to several different problems relating to the stability of the atrial defibrillation threshold (ADT) and the safety of such a technique if used in an automatic mode (7).

Recent experimental studies on animals showed that low energy intracardiac atrial shocks, if well synchronized on the R wave, can in some instances be dangerous because of the possibility of inducing ventricular tachyarrhythmias (8). This phenomenon was observed in the animals when the intracardiac shock was synchronized on the R wave with an RR interval <300 ms (8).

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No human data on this dangerous outcome has yet been published. The aims of this study were to analyze the change in the intracardiac ADT at rest and during exercise; to quantify the effective risk of this procedure in patients with chronic AF in a specific situation, like the effort during which the higher level of circulating catecholamines and the presence of shorter RR intervals could increase the likelihood of generating malignant ventricular arrhythmias; and to compare the ADT of chronic and reinduced AF.

Methods

Subjects and study protocol. Sixteen patients with chronic AF (duration 10 days or more) who were candidates for low energy intracardiac atrial cardioversion were included in the study. The clinical data of the patients are reported in Table 1. The patients' mean (±SD) age was 63.25 ± 9.69 years (range 43 to 78); six of them were men and 10 women; their mean body weight was 71.88 ± 13.56 kg; their mean height was 163.25 ± 9.60 cm; and their mean body surface area was 1.76 ± 0.20 m^2. The mean left atrial diameter was 47.13 ± 7.11 mm. The underlying heart disease was valvular disease in nine patients (56%), hypertension in five (31%) and hyperthyroidism (corrected before the procedure) in two (12%). The mean

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Abbreviations and Acronyms

ADT = atrial defibrillation threshold
AF = atrial fibrillation

duration of atrial fibrillation was 169.38 ± 229.27 days (range 10 to 960). Ten patients were taking antiarrhythmic drugs before the intracardiac cardioversion: eight (50%) amiodarone and two (12%) propafenone (Table 1). All the patients were fully orally anticoagulated for at least 20 days before the procedure and did not present at transesophageal echocardiography with auricular thrombi or spontaneous echo contrast (9,10). Anticoagulant drugs were discontinued at least 2 days before the procedure and begun again immediately after it and continued for 1 month.

The study was approved by our Institutional Ethical Committee, and all the patients gave written informed consent.

Catheter position and shock wave form. Three intracavitary temporary leads were used in each patient. Two 6F custom-made temporary catheters (Electro-Catheter, Inc.) were used for the shock and one USCI tetrapolar lead for ventricular synchronization. The Elecath leads had ten parallel platinum electrodes 4.5 mm long and separated by 2 mm; the active surface area was 314.1 mm^2. The femoral approach was used in all the patients for catheter insertion. The first Elecath lead was positioned under fluoroscopic guidance in the distal part of the coronary sinus to embrace the left atrium as much as possible. The second Elecath lead was positioned in the high right atrium, paying attention to keep the electrodes in contact with the atrial wall. The third lead was positioned in the right ventricular apex (Fig. 1). All the catheters were connected to a Teletronics Guardian implantable defibrillator especially adapted to receive such catheters. The capacitor size of the defibrillator was 150 μF. The defibrillator was used manually instead of automatically. A truncated, biphasic (3 + 3 ms), exponential wave form was used (11,12). Beginning from 50 V, the voltage was increased progressively by 50-V increments until restoration of the sinus rhythm was obtained. All shocks were separated by an interval of at least 1 min. The voltage, both the total and delivered energy and the impedance of each shock were automatically measured by the device. The total energy was reported in the study.

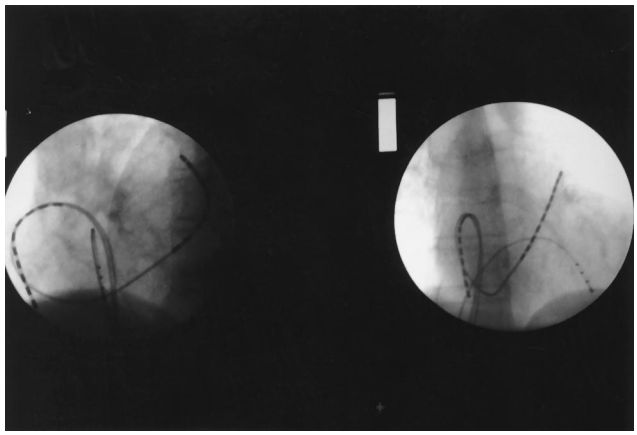


Figure 1. Catheters positioned in the 30° right (**left**) and left (**right**) anterior oblique views before a low energy endocavitary shock. The two decapolar, large, active surface catheters are positioned in the coronary sinus and in the right lateral atrial wall, respectively. The tetrapolar USCI catheter positioned in the right ventricular apex is used for R wave synchronization.

Cardioversion at rest and during exercise. The patients were submitted, in a random order, to two sequences of shocks to determine the ADT—one in the basal condition at rest and

Table 1. Clinical Data of the Patients

Pt No.	Age (yr)/Gender	Height (cm)	Weight (kg)	BSA (m^2)	Underlying Heart Disease	AF Duration (days)	Left Atrial Diameter (mm)	Drug Before DC Shock
1	63/F	164	80	1.86	Valvular disease	10	58	Amio.
2	70/F	168	75	1.84	Hypertension	210	55	Amio.
3	59/F	169	90	2.01	Hyperthyroidism	60	43	None
4	57/F	140	57	1.44	Valvular disease	30	50	None
5	70/M	175	72	1.86	Valvular disease	90	51	Amio.
6	51/F	156	73	1.73	Valvular disease	90	44	Amio.
7	71/F	157	50	1.45	Valvular disease	960	49	None
8	66/F	160	68	1.72	Hyperthyroidism	240	41	None
9	71/M	165	74	1.85	Hypertension	30	29	None
10	43/F	150	58	1.52	Valvular disease	60	48	Propa.
11	73/M	170	62	1.71	Valvular disease	360	58	Propa.
12	48/F	160	58	1.59	Valvular disease	90	49	Amio.
13	61/M	180	105	2.24	Hypertension	150	43	Amio.
14	68/M	168	75	1.84	Hypertension	150	47	Amio.
15	78/M	165	76	1.81	Hypertension	90	46	None
16	63/F	165	77	1.73	Valvular disease	90	43	Amio.
Mean	63.25	163.25	71.88	1.76		169.38	47.13	
SD	9.69	9.60	13.56	0.20		229.27	7.11	

AF = atrial fibrillation; Amio. = amiodarone; BSA = body surface area; DC = direct current; F = female; M = male; Propa. = propafenone; Pt = patient.

one during exercise. Exercise was performed isometrically by the superior limbs and continued until the heart rate increased at least 20% compared with the basal condition. The exercise was repeated for each step of the procedure. After the first restoration of sinus rhythm, either during exercise or at rest, AF was induced again by means of burst atrial pacing after 15 min. The induced AF had to be stable for at least 1.5 min before the initiation of the second series of shocks. About 1.5 min after AF reinduction the cardioversion protocol was repeated until the ADT was reached.

Neither general anesthesia nor sedation was used in any patient. Continuous electrocardiographic monitoring and recording were performed immediately before and during each step of the cardioversion. After each shock the patients were requested to grade the discomfort with a score from 1 to 5 (1 = not felt; 5 = severe discomfort).

At the end of the procedure a blood sample for creatine kinase analysis was taken to discover possible cellular damage.

A transthoracic echocardiogram was performed within 24 h of the procedure to look for pericardial effusion or contractile defects.

Statistical analysis. Data are presented as mean values ± SD. When appropriate, the following tests were performed: the Student paired and unpaired *t* test; two-way repeated measures analysis of variance; multifactorial analysis of variance for repeated measures was adopted for testing the influence of both sequences of exercises (groups A and B) and sequences of shocks (first and second shocks) on the defibrillation threshold. A p value <0.05 was considered significant. The power of the study was >90% in detecting a difference in the ADT of 25% between the groups.

Results

The patients were classified into two groups according to the assigned order of cardioversion: Group A included nine patients who underwent the first intracardiac cardioversion during exercise and the second one at rest. Group B included seven patients who underwent the first cardioversion at rest and the second during exercise.

Successful cardioversion of both spontaneous and reinduced AF was obtained in all the patients. Figure 2 shows an example of intracardiac cardioversion at rest and during isometric exercise in the same patient.

Atrial defibrillation threshold. The principal intracardiac cardioversion data are reported in Table 2. The mean ADT in group A was 7.10 ± 1.07 J (range 5.7 to 8.3) during exercise and 6.42 ± 1.77 J (range 2.5 to 10) at rest (p = 0.38); in group B, the mean atrial defibrillation threshold was 6.19 ± 1.97 J (range 2.5 to 8.3) during exercise and 7.79 ± 0.23 J (range 7.7 to 8.3) at rest (p = 0.06). Regardless of whether the cardioversion during exercise was performed for the first or the second time, considering the total group of patients, the mean ADT was 6.70 ± 1.54 J (range 2.5 to 8.3) during exercise and 7.02 ± 1.82 J (range 2.5 to 10) at rest (Fig. 3). This difference was not significant (p = 0.59).

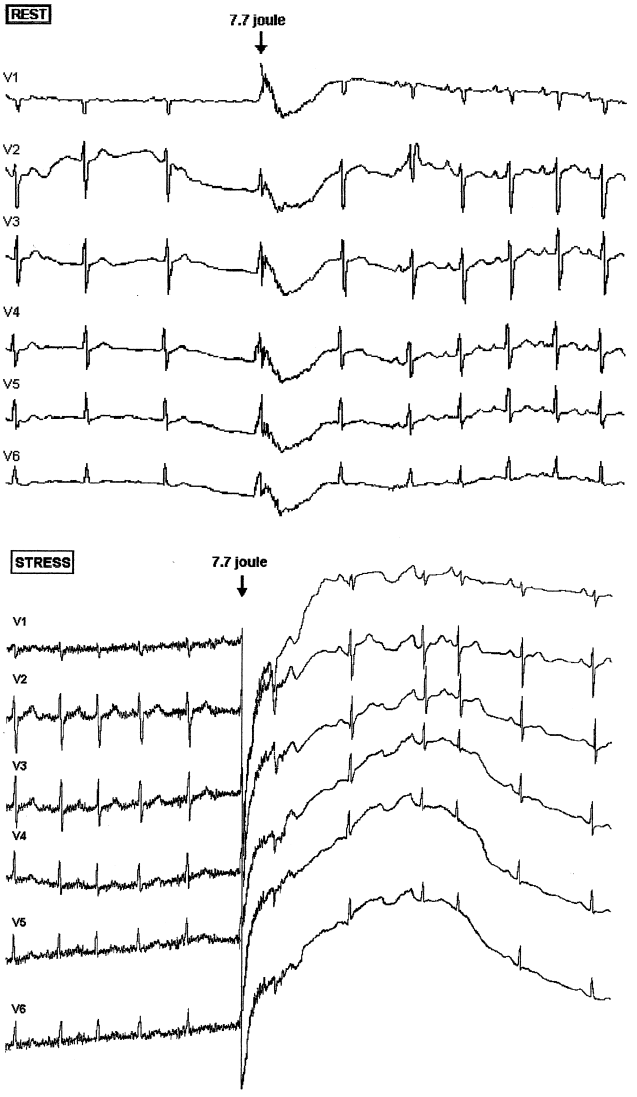


Figure 2. Example of internal cardioversion at rest and during isometric exercise in Patient 9. Chronic AF is converted to sinus rhythm by a 7.7-J shock synchronized on the R wave, both at rest (**top**) and during exercise (**bottom**).

When comparing the first ADT with the second one in the total patient group, regardless of exercise or rest conditions, a significantly lower ADT was observed in the second shock sequence compared with the first (6.32 ± 2.09 J vs. 7.40 ± 0.87 J, $p < 0.05$) (Fig. 4). No difference in the ADT was observed between the mean ADT value in all patients of group A (6.76 ± 1.77 J) (which included three patients on amiodarone and one on propafenone) and that in group B patients (6.99 ± 1.58 J) (which included five patients on amiodarone and one on propafenone) (p = 0.71).

Antiarrhythmic drugs were administered to four patients of group A (amiodarone in three and propafenone in one) and to six patients of group B (amiodarone in five and propafenone in one).

No other significant difference in the ADT appeared, comparing its value during exercise and at rest when deter-

Table 2. Intracardiac Cardioversion Data

		1st Shock		2nd Shock		Rest			Stress		
Pt No.	Random	J	Tol. (score)	J	Tol. (score)	J	Rate	Tol. (score)	J	Rate	Tol. (score)
1	S	5.70	4	6.20	3	6.20	70	3	5.70	120	4
2	R	7.70	3	5.70	3	7.70	120	3	5.70	150	3
3	S	7.70	4	7.70	4	7.70	70	4	7.70	110	4
4	S	7.70	3	4.00	4	4.00	75	4	7.70	100	3
5	R	7.70	3	5.70	3	7.70	100	3	5.70	140	3
6	R	8.30	3	8.30	2	8.30	90	3	8.30	110	2
7	S	5.70	3	2.50	4	2.50	90	4	5.70	110	3
8	S	8.30	4	10.00	5	10.00	110	5	8.30	140	4
9	R	7.70	2	7.70	3	7.70	70	2	7.70	90	3
10	S	7.70	2	5.70	2	5.70	70	2	7.70	110	2
11	R	7.70	2	5.70	2	7.70	90	2	5.70	130	2
12	S	5.70	2	8.30	3	8.30	80	3	5.70	100	2
13	R	7.70	3	2.50	2	7.70	45	3	2.50	70	2
14	S	7.70	3	7.70	4	7.70	70	4	7.70	110	3
15	S	7.70	3	5.70	3	5.70	80	3	7.70	110	3
16	R	7.70	4	7.70	4	7.70	100	4	7.70	120	4
Mean		7.40	3.00	6.32	3.19	7.02	83.13	3.25	6.70	113.75	2.94
SD		0.87	0.73	2.09	0.91	1.82	18.61	0.86	1.54	19.96	0.77

R = rest; S = stress; Tol. = tolerance.

mined with the first series of shocks (7.10 ± 1.07 J vs. 7.79 ± 0.23 J, respectively, p = 0.12) or when determined with the second series of shocks (6.19 ± 1.97 J vs. 6.42 ± 2.29 J) (p = 0.06).

Again, no difference was observed when comparing the exercise ADT during the first sequence of shocks (7.10 ± 1.07 J) with the exercise ADT during the second sequence of shocks (6.19 ± 1.97 J) (p = 0.25), and the rest ADT determined with the first sequence of shocks (7.79 ± 0.23 J) with the rest ADT determined with the second sequence of shocks (6.42 ± 2.29 J) (p = 0.14).

Ventricular rate and RR interval preceding the shocks. The mean ventricular rate was significantly lower at rest (83.13 ± 18.61 beats/min) than during exercise (113.75 ± 19.96 beats/min) (p < 0.0001) (Fig. 5).

The mean RR interval preceding the shock was 622.7 ±

Figure 3. Difference in ADT between shock delivered at rest and during exercise. Data are expressed as individual values (**continuous lines**) and mean values (**thick bars**) ± SD (**thin bars**). See text for explanation.

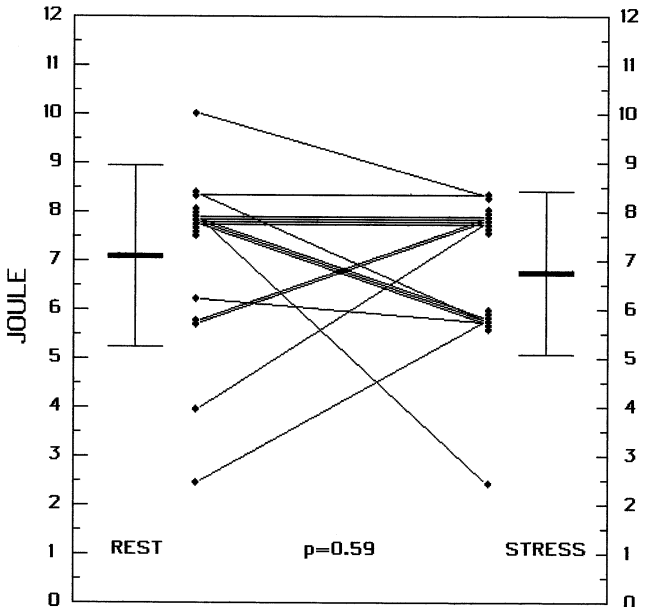
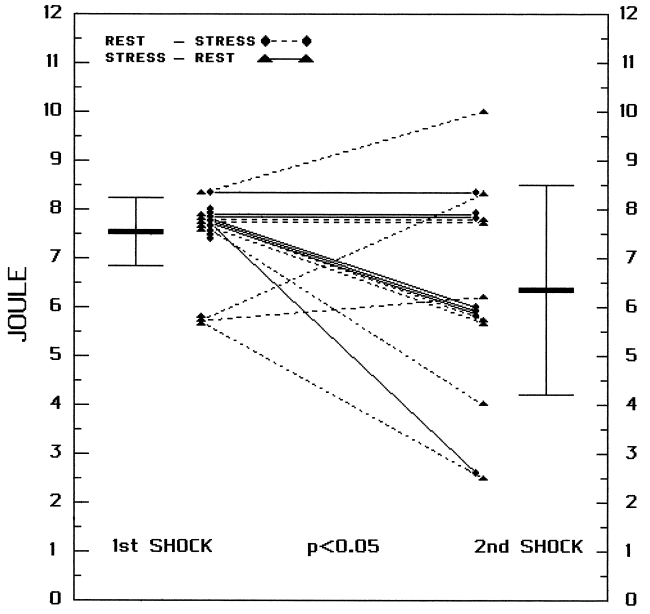


Figure 4. Difference in ADT between the first and second shocks. Data are expressed as individual values (**continuous lines** = rest/stress; **dotted lines** = stress/rest) and mean values (**thick bars**) ± SD (**thin bars**). See text for explanation.



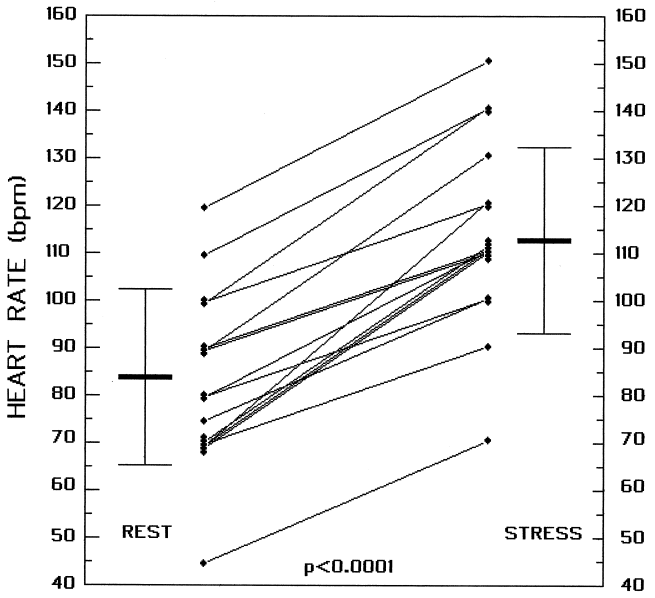


Figure 5. Difference in the ventricular rate at rest and during exercise. Data are expressed as individual values **(continuous lines)** and mean values **(thick bars)** ± SD **(thin bars)**. See text for explanation.

216 ms. Considering all the 161 shocks given in the total group, there was no significant difference between the median RR interval preceding the shocks at rest and during exercise (654.30 ± 228.68 ms and 591.95 ± 218.33 ms, respectively, p = 0.07).

An RR interval preceding the shock <300 and 350 ms was seen only in two and three cases, respectively. Ventricular synchronization was appropriate in all the 161 shocks given. Ventricular arrhythmias were not induced in any of the patients by the intracardiac atrial shocks. As far as patient discomfort is concerned, the average score of the successful shocks was 3.25 ± 0.86 at rest and 2.94 ± 0.77 during exercise (p = 0.09). Comparing the mean discomfort score of all the first sequences of shocks with that of the second ones, no relevant difference was noted (3.00 ± 0.73 during the first and 3.19 ± 0.91 during the second sequence of shocks; p = 0.33). In this group of patients, the discomfort score was not related to the ADT. In fact, considering all the 32 sequences of shocks, the ADT was not different in the five groups identified according to the tolerance score (score 2: 6.38 ± 1.90 J; score 3: 7.11 ± 1.04 J; score 4: 6.56 ± 2.04 J; p = NS between all the groups).

A pericardial effusion or a contractile deficit was not discovered in any of the patients by transthoracic echocardiography performed within 24 h of the procedure. No embolic episodes were observed after the intracardiac cardioversion, nor was a creatine kinase elevation detected in any patient.

Discussion

The possibility of reconverting paroxysmal and chronic AF to sinus rhythm by means of low energy intracardiac atrial cardioversion is a reality (1–4). An implantable atrial defibrillator, alone or in combination with a ventricular defibrillator, will soon be proposed as an alternative to the drug treatment of recurrent AF (5–7). This gives rise to question of ADT behavior during daily life activities and the potential danger of intracavitary atrial shocks to induce ventricular arrhythmias (8). Furthermore, no data are yet available on ADT changes during exercise.

Previous studies on low energy intracardiac cardioversion of AF. Ayers et al. (8) showed, in the sheep, that low energy intracavitary atrial shocks may induce ventricular fibrillation (2 [0.28%] of the 716 shocks given during AF) if the preceding cycle length is ≤300 ms, whereas no episodes of ventricular fibrillation (of 895 shocks) were induced with a preceding cycle length >300 ms. Looking more deeply into the Ayers report (8), we can see that in the sheep model with induced AF, ventricular fibrillation was induced only by 2 of the total 716 delivered shocks. Both these episodes of ventricular fibrillation were initiated with a very short cycle length preceding the shock (198 and 240 ms, respectively).

Murgatroyd et al. (1) recently confirmed, in humans, the safety of low energy intracardiac cardioversion of paroxysmal AF by delivering shocks only after RR intervals >500 ms.

Alt et al. (2) had a 91% success rate in restoring sinus rhythm in 11 patients with chronic AF with a mean energy level of 3.7 ± 1.7 J and without induction of ventricular fibrillation.

Significant differences can be observed among these reports and our study, as the Ayers (8) and Murgatroyd (1) works examined only animals and humans with induced AF and paroxysmal episodes of the arrhythmia, which require much less energy than chronic fibrillation to be converted to sinus rhythm. Furthermore, paroxysmal AF usually has a much higher ventricular rate than the chronic form, and it is therefore more probable to find short RR intervals.

General features of our study. We included only chronic AF, and this may explain the lower mean ventricular rate either at rest or during isometric exercise and the higher ADT. The RR interval preceding the shock in our patient group was <350 ms only in three cases and <300 ms only in two cases; therefore, nothing can be stated by this data about the real risk of inducing ventricular fibrillation in patients with frequently very short RR intervals. Considering chronic AF, low energy intracardiac shocks proved to be very effective and safe either at rest or during exercise. In our study the ventricular rate increased significantly during exercise, ~33%, reaching a mean value of 113.13 ± 20.56 beats/min (Fig. 5).

This means that circulating catecholamines were increased significantly (13) and that the shocks were given in a situation potentially at more risk to induce ventricular arrhythmias (14,15). Nevertheless, in none of the patients was ventricular arrhythmia induced by the intracavitary shocks.

Difference in ADT in chronic and reinduced AF. As far as the ADT is concerned, it appeared clear by this study that the second sequence of shocks—that is, the intracavitary cardioversion of the reinduced atrial fibrillation—had a significantly lower ADT (6.32 ± 2.09 J) than the first sequence of shocks

(7.40 ± 0.87 J) ($p < 0.05$) (Fig. 4), regardless of exercise or rest conditions. This reduction of the ADT can be interpreted as the result of the new electrophysiologic situation created by the interruption of the chronic atrial arrhythmia and the creation of a new induced arrhythmia, which could be considered halfway between a chronic and a paroxysmal form of AF. In fact, compared with chronic arrhythmia, the ADT of the reinduced arrhythmia is, as said earlier, lower. Nevertheless, it still remains much higher than the ADT usually reported for the spontaneous paroxysmal episodes (1) or for the pacing-induced form of AF in patients or animals (16,17) without previous atrial arrhythmias. It is probable that the anatomic or at least the electrophysiologic substrate of induced AF in patients recently reconverted to sinus rhythm from chronic AF is significantly different from that of similarly induced or spontaneous paroxysmal AF in a subject usually in sinus rhythm for long periods of time. Our results are also in agreement with the study of Wijffels et al. (18). These investigators showed that the electrophysiologic changes related to the development of sustained AF return to normal values with time after cardioversion. In their study, after cardioversion of long-lasting AF, a prolongation of the effective atrial refractory period and a lengthening of the local fibrillation interval in reinduced AF were found. This suggests that reinduced AF is less complex, more organized and probably sustained by a lower number of electrical circuits than the persistent form. This could account for the lower ADT of the reinduced AF in our study. Moreover, Alt et al. (19) showed a more marked decrease in the ADT from initial to subsequent cardioversion, but over a much longer period (months).

Effect of exercise on ADT and tolerance score. As far as the effect of exercise on the ADT is concerned, we could not find any significant change in the shock energy necessary to restore sinus rhythm when compared with the rest ADT. Therefore, the increased circulating catecholamines do not seem to influence the ADT, but only the atrioventricular conduction (20). However, there is an apparent trend toward decreased defibrillation thresholds during exercise, which could become significant by extending the study to a larger number of patients.

The discomfort score given by the patients did not change significantly between the two sequences of shocks, at rest and during exercise, or between the first and the second sequence of shocks. The procedure did not have to be stopped in any of the patients because of patient intolerance.

Conclusions. The restoration of chronic AF by means of low energy intracardiac cardioversion is feasible and carries a success rate of almost 100%. The patients tolerate the procedure sufficiently well without sedation or general anaesthesia, both at rest and during exercise. The procedure used in patients with chronic AF is safe both at rest and during exercise. The ADT of reinduced AF is lower than that of chronic AF.

Further studies are needed to clarify the risk of low energy intracardiac cardioversion of paroxysmal AF during exercise.

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